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Histological Alterations in the gills of *TILAPIA MOSSAMBICUS* exposed to Kerosene

Gayathri V[1], Dr.Mazher Sultana[2]
1 Department of Biotechnology, Rajalaksmi Engineering College, Thandalam, Chennai, INDIA
2 Department of Advanced Zoology and Biotechnology, Presidency College, Chennai, INDIA
gopigaya9@rediffmail.com, sultanaafzal@yahoo.com

Abstract:
PAHs are rapidly accumulated by aquatic organisms reaching level higher than those in the ambient medium and affecting normal functions of the aquatic life. Kerosene is a well-known source of harmful compound including PAHs. Histopathological investigations have been proved to be a sensitive tool to detect effects of chemical compounds within target organs of fish in laboratory experiments. In this study it was proposed to find out whether exposure to kerosene for 96 hrs has an effect on the gills of *Tilapia mossambicus* because, gills are the first organ and main target for many aquatic pollutants. Varied morphological changes occurred in the gills of *Tilapia mossambicus* treated with kerosene. The gill exhibited marked alterations in their epithelia. The epithelium was no longer continuous, particularly the more delicate respiratory lamellae. Thus, there was fusion at adjacent secondary lamellae as a result of hyperplasia. Oedema at the secondary lamellae and swelling of the epithelial cells were observed. The pillar cells have been altered and blood spaces expanded. There is a severe hyperplasia with profound oedematous changes characterized by epithelial detachment. The elongated secondary lamellae with club-like structures were the other pathological changes observed in the histological sections of gills of *Tilapia* treated with water soluble fraction of kerosene. the number of lesions caused by kerosene is related to its concentration. Thus the result of these studies clearly indicates that PAH has diverse effects on the gills.

Keywords: Environmental pollution, Tilapia mossambicus, Kerosene, Gills, Histopathology.

INTRODUCTION

Polycyclic aromatic hydrocarbons [PAHs] are the most toxic components in crude oil, and their percentage increases during the refining process. Kerosene as one of the intermediate distillate products are well-known source of harmful compounds including PAHs. Generally PAHs are less sensitive to photooxidation and therefore, are more persistent in water. PAHs are rapidly accumulated by aquatic organisms reaching level higher than those in the ambient medium and affecting normal functions of the aquatic life [1].

The fish, as a bioindicator species, plays an increasingly important role in the monitoring of water pollution because it responds with great sensitivity to changes in the aquatic environment [2]. Histopathological studies have been conducted to help to establish causal relationships between contaminant exposure and various biological responses. Histopathological investigations have also been proved to be a sensitive tool to detect direct effects of chemical compounds within target organs of fish in laboratory experiments [3,4]. The fishes get their tissues easily damaged due to water pollutants, the gills and liver being the most potent sites that show changes in their histoarchitecture at very early stages of toxic stress [5]. This study focused on gills because they are the main target for many aquatic pollutants in general, and one of the most seriously affected organ due to direct contact with the aquatic environment [6]. In fish, gills are the first organ to which any pollutant comes into contact. Fish gills are very sensitive to changes in the composition of the environment and are an important indicator of waterborne toxicants. Consequently, injury to gill epithelium is a common response observed in fish exposed to a variety of contaminants. The severity of damage to the gills depends on the concentration of the toxicant and the period of exposure.

MATERIALS AND METHODS

Healthy *Tilapia mossambica* weighing 20–25 g of both sexes were collected from fresh water and fed once a day with 1.5% body weight commercial fish feed. The fish were also maintained at 12L: 12D cycle with a water temperature of 30˚C. The fish were acclimated to these conditions for at least two weeks before the experiment. Feeding was stopped 24 hr prior to kerosene exposure. Fishes were exposed to the water soluble fraction of kerosene and LC_{50} value was calculated by Finney method. Then the experimental fishes were treated with different subleathal concentration of kerosene [75, 150, 300 ppm]. Simultaneously separate control fish was maintained. Each experiment was set up in triplicate. The exposure lasted for 96 hrs. The histopathological observations were made from gills of the control and experimental groups and were fixed in 4% neutral buffered formalin. The fixed tissues were dehydrated in an increasing gradient of alcohol [70, 80, 90, and 100%] for 30 min each and were eventually dried in acetone, and cleared in xylene for 30min. The tissues were then infiltrated by embedding in molten wax and sectioned at 8µ. The paraffin sections were then mounted on a slide, stained with haematoxylin and counterstained with eosin [7].

To study the pathogenicity of various concentrations of toxicant, the section of gills were observed under microscope and the condition in different tissues were photographed at lower and higher power of magnification using Nikon micro photographic equipment.

RESULT AND DISCUSSION

Histology is a useful technique for investigating the toxic effect of various pollutants. Such a study also offers opportunity to locate the effect of pollutants in various organs and systems of animals. This type of study in fish has been handicapped to a great extent because of the lack of adequate histological literature concerning various fish organs. Considerable interest has been shown in recent years in histopathological studies while conducting sub-lethal tests in fish. Tissue changes in test organisms exposed to sub-lethal concentration of toxicant are a functional response of organism which provides information on the nature of toxicant.

There is considerable information indicating that pesticides and heavy metals are responsible for many adverse effects in fishes and other animals from the histopathological and histochemical points of view [5]. The chemical variables influence toxicity by affecting the respiratory rate. As the gills are the respiratory and osmoregulatory organ in fish, any damage occurring in the tissue may impair respiration. Visible histological abnormalities caused due to toxicity of heavy metal in animals have been reported in earlier studies [8, 9, 10 and 11]. The exposure of fish to pollutants is likely to induce a number of lesions in different organ like gills, kidney and brain. The exposure of aquatic organisms to very low level or sub-lethal concentration of pesticides in their environment may also result in various hisotopathological alterations in vital tissues. Behavioral disturbances, hepatic and renal injury were observed in the white fish [*Coregonus clupeaformis*] exposed to nickel [12] and lake trout [*Salvelinus namaycush*] exposed to lindane [13].

The gill is an important site for the entry of toxicants that provoke lesions and gill damage [11]. *Tilapia mossambicus* exposed to toxicants showed an extensive damage to their gill architecture in the present study and this is in agreement with the earlier observations [14, 15]

Varied morphological changes occurred in the gill tissue of the water soluble fraction of kerosene treated *Tilapia mossambicus* and the gills exhibited marked alterations in their epithelia (Fig:1). The epithelium was no longer continuous, particularly the more delicate respiratory lamellae. Thus, there was fusion at adjacent secondary lamellae as a result of hyperplasia. Oedema at the secondary lamellae and swelling of the epithelial cells were observed. The pillar cells have been altered and blood spaces expanded. There is a severe hyperplasia with profound oedematous changes characterized by epithelial detachment. The elongated secondary lamellae with club-like structures were the other pathological changes observed in the histological sections of gills of *Tilapia mossambicus* treated with water soluble fraction of kerosene (Fig: 1.b, 1.c, 1.d). The general morphological changes in the gills recorded when compared to control fish (Fig: 1.a) in the present study have also been reported in *Astyanax* sp. after 96 hr brief exposure to water soluble fraction of crude oil [16] and *Clarias gariepinus* under a brief or prolong exposure to plant extract [17,18].

The changes in the gills were adaptation by the fish to cope up with challenge of the toxicant. For example atrophy or dystrophy, vacuolation, degeneration, curving, clubbing and fusion of the secondary lamellae were attempts by the fish to reduce available surface area to the kerosene (Fig-1.b, 1.c, and 1.d). But this may result in the reduction of available surface for respiration and ionic exchange, consequently resulting in an internal hypoxic condition under toxic environment. Oedema recorded in the gills was due to failure of sodium pump occasioned by the toxicant leading to accumulation of Na^+ and the diffusion of K^+ outside [19]. Vascular changes in gills of exposed fish could be an attempt by the fish to supply more blood to the gills to increase oxygen uptake and supply to the internal organs.

The results of the present study point to the fact that the toxins of pollutant definitely affect the aquatic life of the fresh water fish. The problem can be more serious in the fish surviving in the lake, which were already loaded with toxic residues of all kind. Added to this if the fresh water used is also equally polluted with contaminants and pathogenic flora and fauna, the problem becomes compounded, with simulated experimental conditions as in the laboratory. Hence, a scientific method of detoxification is essential to improve the health of these economically important fish and reduce the losses caused by anthropogenic stress.

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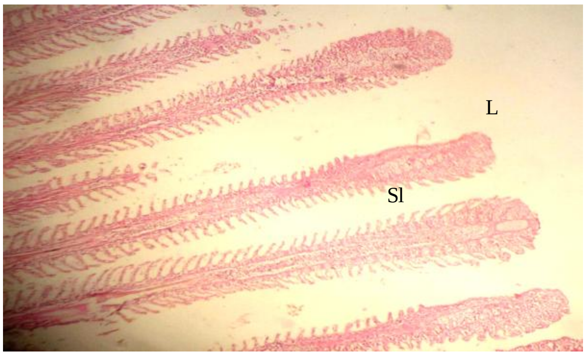
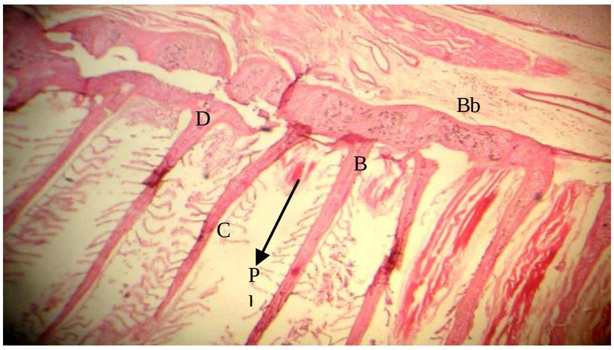
1.A Control



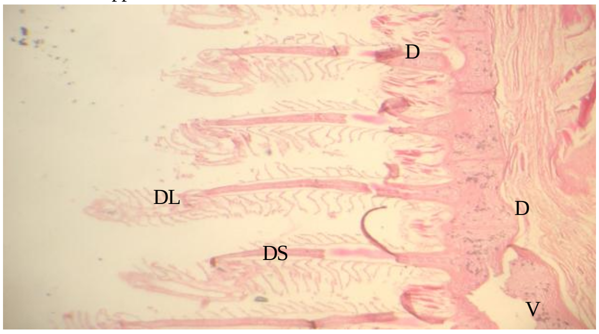
Lt- Lamellar tip SL- Secondary Lamella

1.B 75 ppm



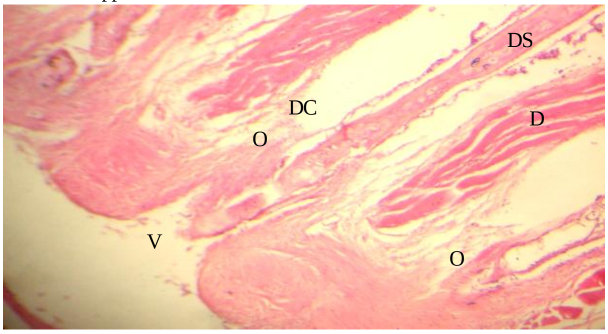
PL- Primary Lamella CL- Curved Lamella
BV- Blood vessel D- Degeneration

1.C 150 ppm



D – Degeneration V- Vacuolation
DLt- Degenerated lamellar tip
DSL- Degenerated secondary lamella

1.D 300ppm



O- Oedema V- Vacuolation
D- Degeneration
DSL- Degenerated secondary lamella
DCc- Degenerated Chloride cells

Fig: 1 Showing the Histology of control and treated Gills of *Tilapia mossambicus*

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